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Guidelines for Surface Water Quality

Vol. 1 Inorganic Chemical Substances

CADMIUM

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INLAND WATERS DIRECTORATE,
WATER QUALITY BRANCH,
OTTAWA, CANADA, 1979.

(Résumé en français)

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Abstract

A literature survey was carried out on the toxic effects of cadmium and cadmium compounds on human health, aquatic life, plants and livestock. The information is summarized in this publication. From it, maximum cadmium concentrations in water at which toxic effects will not appear are recommended.

Résumé

On a effectué une étude de la documentation portant sur les effets toxiques du cadmium et de composés du cadmium sur les humains, les organismes aquatiques, les plantes et le bétail. Ces données sont résumées dans cette publication. En se basant sur ces données, on propose des valeurs des concentrations maximales de cadmium dans l'eau auxquelles on n'observe pas d'effets toxiques.

Acknowledgments

The Water Quality Objectives and Standards Group greatly appreciates the assistance of many dedicated scientists located throughout Federal Agencies, especially Health and Welfare Canada, Agriculture Canada, National Research Council of Canada and Environment Canada.

Special thanks are expressed to K.W. Taylor and E. Brien of the Water Quality Branch, who assisted directly in the production of this publication.

RECOMMENDED WATER QUALITY OBJECTIVES FOR CADMIUM

Uses	Recommended objectives
Raw public water supply	0.01 mg/L as total cadmium for waters with conventional treatment; 0.02 mg/L as total cadmium for waters with conventional plus lime precipitation–activated carbon treatment
Aquatic life and wildlife	
Aquatic life	0.0002 mg/L as total cadmium
Wildlife	Insufficient information
Agriculture water supply	
Livestock watering	0.02 mg/L as total cadmium
Irrigation water	0.01 mg/L as total cadmium
Recreation and aesthetics	
Recreational waters	0.01 mg/L as total cadmium
Industrial water supplies	
Food processing	0.01 mg/L as total cadmium

Guidelines for Water Quality
Vol. 1. Inorganic Chemical Substances. Cadmium

Stewart W. Reeder, Adrian Demayo and Margaret C. Taylor

SOURCES, OCCURRENCE AND FORM

Natural Distribution of Cadmium

In the list of the elements in the earth's crust, cadmium ranks sixty-fourth in order of abundance with an average concentration of 0.2 mg/kg (Taylor 1964), which, for example, is less than that of chromium, zinc, copper and lead. The average cadmium concentration in sea water is 0.00011 mg/L, placing Cd forty-third in order of abundance (Bowen 1966, pp. 19-20).

There are no known commercial deposits of cadmium. Cadmium commonly occurs as greenockite (CdS), which is found associated with zinc sulphide ore, particularly sphalerite, (Zn, Fe)S (Brown 1977). Canadian zinc ores contain up to 0.07% cadmium (Barry 1975).

Production and Uses of Cadmium

Cadmium is obtained as a by-product of zinc refining due to their close association. Cadmium recovery from zinc ore ranges from 31.5% to 67.5% (70% to 90% from ore to concentrate and 45% to 75% from concentrate to metal). Cadmium is recovered from the fumes produced during the roasting of zinc ores and concentrates and from the precipitates obtained during the purification of zinc sulphate solution (Brown 1977).

Owing to its nature, the production of cadmium depends not on the demand for cadmium but on the production of zinc. Secondary sources of cadmium are negligible in terms of total supply. The production of cadmium metal in the Western World in 1975 was 11 226 tonnes compared with 13 550 tonnes in 1974, a decrease of approximately 17%. Japan was the major producer of cadmium in 1975 with 2547 tonnes (22.7% of the total), followed by the United States with 1990 tonnes (17.7%) and Canada with 1290 tonnes (11.5%). The Canadian production of cadmium in 1975 represented a slight increase, 3.3%, over the 1974 production. At the same time, however, it was considerably lower than the level of 2365 tonnes reached in 1969 (Brown 1977).

The estimated 1975 Western World consumption of cadmium, 8532 tonnes, represented a drop of 38% from the 1974 level of 13 760 tonnes. The major consuming countries are the United States, Japan, Germany and the United Kingdom. The estimated Canadian consumption of cadmium in 1975 was only 30 tonnes, while the 1974 consumption was 48 tonnes.

Cadmium is used mainly in electroplating other metals or alloys for protection against corrosion and in the manufacture of pigments in the yellow-to-orange series (cadmium sulphides) and the pink-to-red and maroon series (cadmium sulphoselenides). Other cadmium compounds are used in the production of PVC (cadmium stearate) and tubes for television sets (cadmium phosphors). Cadmium metal is also used in cadmium-silver solders; in telephone and trolley wires (1% cadmium and the rest copper); in metal sheets for automobile radiator fins, nickel-cadmium batteries, valve seats for high-pressure gas containers; and in low melting point alloys for automatic sprinkler systems and fire detection apparatus.

The distribution of cadmium consumption among various industries in the United States is: electroplating, 55%; plastics, 20%; pigments, 12%; Ni-Cd batteries, 5%; and miscellaneous, 8% (Yost 1978).

Pathways for Entering the Environment

The transfer and conversion of cadmium in the different compartments of the ecosystem are schematically represented in Figure 1.

Bertine and Goldberg (1971) estimated that approximately 500 tonnes of cadmium enter the environment yearly as a result of natural weathering phenomena.

The main anthropogenic sources of cadmium in the environment are (i) the emissions to air and water from mining, metal (zinc, lead, copper) smelteries and industries involved in the manufacture of alloys, paints, batteries and plastics; (ii) the use of sludges, fertilizers and pesticides containing cadmium in agriculture; and (iii) the burning of fossil fuels (Friberg *et al.* 1974, p. 9).

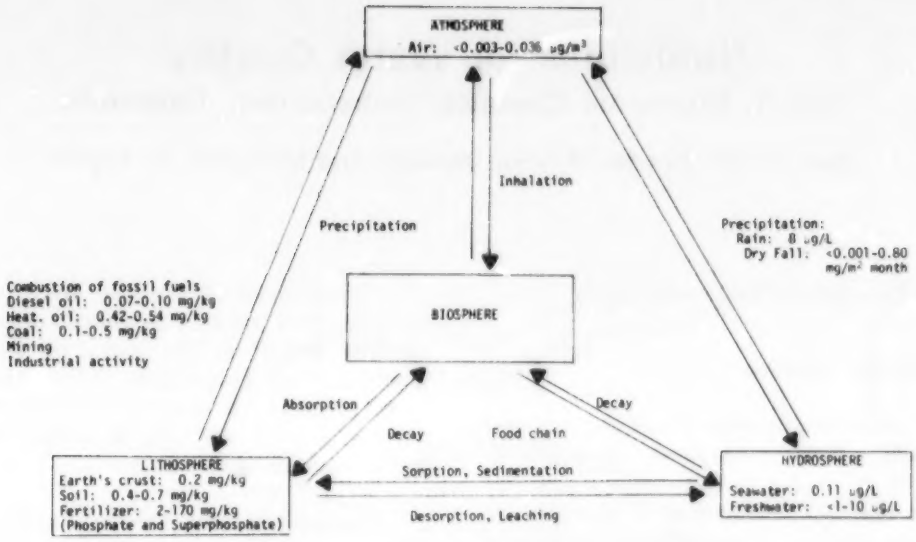

Figure 1. Cadmium transfer and conversion in the ecosystem (Fishbein 1974).

The total anthropogenic cadmium emissions in the environment in 1968 in the United States alone have been estimated at 2500 to 3600 tonnes. Of the estimated loss, 160 to 1230 tonnes came from the burning of fossil fuels, use of fertilizers, etc., i.e. those activities not directly related to cadmium use (Goeller *et al.* 1973, p. 87). The difference between the consumption and direct losses of cadmium, approximately 2600 to 4800 tonnes, is deposited in landfills, dumps, etc. (Goeller *et al.* 1973, p. 94). Yost (1978), in a simulation of cadmium flow in the United States, calculated that of the cadmium consumed over a 10-year period, 79% is deposited as landfill, 3.5% becomes sewage sludge, and 0.5% dissipates in natural waters.

Emissions to the atmosphere are the major man-made pathway by which cadmium enters the environment. In the United States it is estimated that the air emissions of cadmium can represent almost 75% of the total cadmium losses to the environment (Sittig 1975, p. 32). Among the major cadmium air pollutors are such industries as iron and steel (blast furnace), primary zinc production (especially the roasting phase), and primary copper production. Due to the low cadmium concentration of petroleum and coal, 0.01 and 0.002 mg/kg respectively, the burning of fossil fuels does not seem to be a major source of cadmium to the environment (Bertine and Goldberg 1971).

An experiment following the transfer of cadmium from the terrestrial to the aquatic ecosystem showed that a high percentage (94% to 96%) of the cadmium remained in the soil. The cadmium that reached the water accumulated more rapidly in the sediments than in living organisms. Twenty percent of the cadmium in the water was found on suspended particles (Huckabee and Blaylock 1974).

A study which followed the fate of cadmium entering a pond from an effluent containing approximately 2.0 g Cd/day also indicated that a higher proportion of cadmium was in the sediments and very little was taken up by the aquatic organisms (Lisiecki and McNabb 1975).

Forms of Cadmium

The main form of cadmium is Cd(II), which makes up a series of inorganic (halides, oxide, sulphide, etc.) and organic (dialkyl and diphenyl) compounds. Several Cd(I) compounds have also been reported. Cadmium forms a series of six-and four-coordinate complexes (Aylett 1973, p. 254). Cadmium carbonate, sulphide and hydroxide have very low solubilities and this is probably why most of the cadmium in the aquatic environment is found in the sediments.

In surface waters from the Ottawa region (Rideau Canal, Ottawa River), Ramamoorthy and Kushner (1975) found part of the cadmium as free ion and part bound to compounds with molecular weights between 1400 and 16 000. In the Brewery Creek water, characterized as an "open sewer," most of the cadmium was bound. It was suggested that the binding components were humic acids, which can have molecular weights ranging from 300 to >200 000. It was also suggested that bound Cd(II) is biologically unavailable. Organic ligands reduce the toxicity of Cd(II) to fish and higher animals (Fleischer *et al.* 1974; Friberg *et al.* 1974).

So far no organic compounds of cadmium have been found in the environment. It has been reported that cadmium bonds to tissue proteins of plants and animals (Casterline and Yip 1975; Colucci *et al.* 1975), although only one such protein, metallothionein, has been positively identified (Friberg *et al.* 1975, pp. 4-31).

RATIONALE

Raw Public Water Supplies

Cadmium in the Human Body

Cadmium enters the human body through inhalation and ingestion of food. Although no exact figures are available, it has been estimated that a normal daily diet contains 4 to 64 μg of cadmium (National Environmental Research Center 1975, p. 6-1). Other estimates show much higher values for the daily intake: 200 to 400 μg Cd (Nilsson 1970, p. 23). Lauwerys (1978) quotes data showing daily intakes of 5-80 μg (average 40 μg) via food and 2-10 μg (average 3 μg) via water. In addition, from almost nil to approximately 28 μg Cd/day can be deposited in the respiratory tract of a person according to whether or not the person smokes, the amount smoked, the place of residence and the place of work.

The amount of cadmium retained by the body depends on the intake route. Approximately 60% to 65% of the cadmium deposited in the lungs is absorbed (Lauwerys 1978), but only 1% to 2% or, according to some authors, 5% to 7% (Friberg *et al.* 1975, pp. 4-6) from the normal daily diet is retained in the body, the rest being excreted via feces and urine. By applying these absorption rates to the daily cadmium intake, it was calculated that the amount of cadmium absorbed by adults can range from approximately 0.4 μg for a non-smoker in a rural, urban or industrial area to 23.6 μg/day for a smoker in an industrial area (Lauwerys 1978).

The elimination rate of the part retained in the body is very slow; the biological half-life has been estimated at 10 to 30 years (Friberg *et al.* 1974, p.88). The result is a net accumulation of cadmium in the human body. Experiments with laboratory animals have shown that the biological half-life varies with the cadmium dose. The biological half-life of plasma cadmium and tissue cadmium in rabbits and tissue cadmium in mice was shortened when the cadmium doses were increased (Nomiyama *et al.* 1978).

In organs, cadmium is bound mainly to metallothionein, a low molecular weight protein. At birth, the human body does not contain any cadmium, while an adult body contains 10 to 60 mg (WHO 1978). The kidney stores approximately 35% of the accumulated cadmium and the liver, approximately 15% (Friberg *et al.* 1974, p. 88).

Some chelating agents modify the excretion and absorption rates of cadmium and its distribution in the animal body. Of particular interest with respect to water quality are the effects of EDTA (ethylenediaminetetraacetic acid) and NTA (nitrilotriacetic acid). In studies with rats, both compounds led to higher excretion rates of cadmium via the urine and to lower concentrations in the liver and kidney (Friberg *et al.* 1974, pp. 52ff.). In a marine situation, NTA was shown to decrease the toxicity of cadmium (Eisler *et al.* 1972).

Vitamin B_6 (pyridoxine) increased the cadmium concentration in the kidney and liver of rats and also increased its retention time (Stowe *et al.* 1974).

Toxic Effects of Cadmium to Humans and Laboratory Animals

The acute toxicity which follows a relatively high cadmium ingestion is characterized by severe nausea, salivation, vomiting, diarrhea, abdominal pains, myalgia and weakness. The acute lethal dose by ingestion (a single dose resulting in death) has been estimated to be between 5 and 50 mg/kg of body weight. The emetic threshold has been estimated at 0.04 to 1.3 mg/kg of body weight (Hise and Fulkerson 1973, pp. 239 f.). In experiments with rats, the 14-day LD_{50} values ranged between 130 and 180 mg/kg of body weight. The wide ranges probably reflect the different toxicities of various cadmium compounds (Lorke 1978). In short-term exposure to elevated levels, cadmium will concentrate in the liver followed by subsequent relocation in the kidney (Dugdale 1978; WHO 1978). Repeated exposure to large amounts of cadmium leads to high concentrations in both organs, i.e., in the liver and kidney, up to a limit of approximately 200 to 300 mg/kg (wet weight) in the renal cortex after which urinary excretion of cadmium increases and morphological changes can be detected (WHO 1978).

The most common findings in people exposed to cadmium while working in industry, as quoted by Dugdale (1978), are pulmonary damage, especially emphysema, and renal tubular damage which may eventually cause bone disease, especially in calcium-deficient subjects.

The chronic toxicity associated with long-term effects of low levels of cadmium intake results in total or partial loss of smell, coughing, difficulty in breathing, weight loss, yellowing of teeth, caries, generalized irritability, gastrointestinal upset, low level of blood hemoglobin, and proteinuria. Experimental and other evidence suggest that cadmium also has carcinogenic, genetic, teratogenic, testicular and cardiovascular effects (Smith 1973, pp. 275 ff.; Friberg *et al.* 1974, pp. 128 ff.; Friberg 1976, pp. 135 ff.; Lorke 1978). Friberg (1978) quoted data showing that

teratogenic effects were observed in experiments with mice after long-term exposure to 10 mg Cd/L in water. Data quoted by Friberg (1976, p. 148) showed that cadmium at a level of 50 mg/L in the drinking water decreased calcium absorption and led to histopathological changes in the duodenum of rats.

Lauwerys (1978) quoted a daily absorption of 10 to 12 μg of cadmium during the whole life span as a rough estimate of the threshold effect level. Depending on the percent absorption this means an ingestion of approximately 0.14 to 1.2 mg/day.

In a Japanese study, a cadmium concentration in rice of under 0.30 to 0.40 mg/kg has been identified as the threshold value for avoiding renal damage in people over 50 years of age (Nogawa 1978). Cadmium (as $CdCl_2$) levels of up to 30 mg/kg of diet were tolerated without any toxic effects by rats and dogs. Doses of up to 100 mg Cd/kg of diet did not reduce the fertility or the rearing capacity during the weaning period of rats. Only very slight toxic effects were observed in a two-year study with rats fed a diet containing 50 mg Cd/kg (Lorke 1978). A dose level of approximately 6 mg Cd/kg of body weight (as $CdCl_2$) which was administered daily for 10 days by stomach tube reduced weight gain and changed the behaviour of pregnant rats, while a dose of 1.8 mg Cd/kg of body weight did not have any effect (Lorke 1978). Rabbits fed a diet containing 300 mg Cd/kg showed enzymuria and aminoaciduria at the sixteenth week of the experiment (Nomiyama *et al.* 1978).

Although questioned by some (e.g., Kitamura 1978; Takeuchi 1978), cadmium has also been pointed out as the main etiological factor in the "Itai-Itai" disease (osteomalacia) first reported in Japan in 1955 (Friberg 1976, pp. 147 ff.; Nogawa 1978; Shigematsu 1978). In the region of Japan where the Itai-Itai disease has been identified, the intake of cadmium has been estimated at 0.6 mg/day or 10 times the amount of a normal Japanese diet (Friberg *et al.* 1974, p. 157). Osteomalacia can be induced by a combination of cadmium exposure and calcium deficiency, but the mechanism is still unclear. Other factors such as malnutrition, pregnancy and aging are also very important in the Itai-Itai disease (Friberg 1976, p. 148).

The kidney is considered to be the critical organ in chronic cadmium poisoning, although the metal accumulates in several body components between which redistribution takes place (Friberg *et al.* 1974, p. 79). The renal cortex has the highest cadmium concentration; in persons with a normal diet, values range from 20 to 50 mg/kg in the United Kingdom, United States and Sweden, and up to 125 mg/kg in Japan. The corresponding blood concentration is below 10 μg/L. In exposed persons, the kidney concentration goes up to 300 mg/kg (although some normal values have also been reported in persons showing signs of cadmium poisoning) and the blood concentrations range between 10 and 100 μg/L (Friberg *et al.* 1974, p. 88). It is interesting to point out that a person with severe renal damage will show a lower cadmium concentration in the renal cortex than persons without or with only slight renal impairment (Friberg *et al.* 1974, p. 107). It has been estimated that 200 mg/kg in the renal cortex is the critical concentration for humans (Friberg *et al.* 1974, pp. 197 ff.), although it has been pointed out that this value seems somewhat arbitrary and, in any case, it is very difficult to determine (Nomiyama *et al.* 1978).

Kjellström (1978), using a series of assumptions, calculated that a cadmium intake of only 24 to 32 μg/day led to a kidney level of 200 mg/kg in 0.1% of the population at age 50. An intake of 44 to 60 μg/day would affect 1% of the population at age 50, and so on.

Using a one-compartment logarithmic accumulation model for cadmium in the human renal cortex and assuming a 4.5% retention of the daily ingestion, a daily elimination of 1% of the body burden (corresponding to a biological half-life of 19 years), it was calculated that with a daily ingestion of 350 μg of cadmium, the critical level in the renal cortex would be reached in 50 years (Friberg *et al.* 1974, pp. 197 ff.).

Modes of Action

Cadmium has an affinity for sulphur and carboxylate sites (Carty *et al.* 1976) and has been shown to inhibit several enzyme systems such as those involved in intermediary metabolism. Cadmium may affect detoxification processes in mammalian liver, e.g., the microsomal NADPH – cytochrome c reductase—and this could increase the toxic effects of other substances (Nilsson 1970). Mills (1974) reported that cadmium lowers the copper content of liver in lambs.

It has been reported that cadmium reduces the iron content of liver and blood plasma and that this can be prevented by administration of vitamin C (Fox and Fry 1970).

Interaction of Cadmium with Other Metals and Compounds

Cadmium is always found in nature in association with zinc and the compounds of the two metals are similar. Zinc has been shown to have a protective effect due to an antagonism towards cadmium in mammals and birds. Necrosis of rat testes, inhibition of growth in chickens and abnormalities of hocks and feathers caused by the cadmium

ion could be prevented by simultaneous administration of zinc. An antagonistic relationship existed between zinc and cadmium with respect to teratogenic effects of large subcutaneous doses of cadmium to pregnant hamsters. Most of the studies used doses which were much higher than would normally be encountered (Ferm and Carpenter 1967). Some of the acute and chronic effects (anemia, weight loss) of cadmium can be prevented by simultaneous application of zinc (Smith 1973; Carty *et al.* 1976).

Zinc is one of the essential elements of life and many enzymes are zinc dependent. Cadmium has a tendency to replace zinc in certain enzymes, altering their stereostructure, impairing their catalytic activity, and causing disease syndromes (Fishbein 1974; Friberg *et al.* 1974, p. 52; Eichhorn 1975). Some symptoms of cadmium poisoning are the same as those produced by zinc deficiencies (Smith and Huckabee 1973). The reverse process, zinc replacing cadmium, is not possible due to the strength of the cadmium protein bond (Cotzias *et al.* 1961).

The effect of zinc is also believed to be partly dependent on the induction of an increased synthesis of metallothionein-like proteins or metallothionein. The teratogenic effect of cadmium has been prevented by simultaneous administration of zinc (Friberg 1976, p. 217). Metallothionein protects against cadmium toxicity and affects the retention and distribution of cadmium in the body (Friberg 1976, p. 137). Metallothionein is composed of 9% metal and 91% thionein, which has 8.5% sulphur in the form of cysteine. It was suggested that cadmium is bound to the protein by an interaction of three sulphydryl groups per metal atom (Smith 1973, p. 273). Metallothionein has been shown to be synthesized in response to the uptake of a toxic cation. With doses of cadmium below a certain critical level, the content of metallothionein in the liver at a given time increased roughly in proportion to the dose (Webb 1975, p. 178).

There is an apparent antagonism of zinc towards cadmium in animals in the ability to complex with amino and carboxylate sites. If both metals compete for the same sites, the toxic effects of cadmium may be expected to be less in the presence of the less toxic zinc (Carty *et al.* 1976).

Recommended Objective for Public Raw Water Supply

Cadmium enters the human body in a variety of forms and from a variety of sources. For nonsmokers, the main source is food and for smokers, the cigarette. Populations in particularly exposed areas (near zinc smelteries, for example) may also inhale significant amounts of cadmium with the air they breathe. The U.N. Food and Agriculture Organization and the World Health Organization have recommended a tolerable weekly intake of cadmium of 0.4 to 0.5 mg or 0.06 to 0.07 mg/day. Accepting the FAO/WHO recommendation and permitting the drinking water to contribute a maximum of 30% of this intake, then the maximum permissible concentration in drinking water should not be greater than 0.01 mg/L of total cadmium (assuming that a person drinks 1.5 to 2.0 L of water per day). This is also the maximum permissible concentration for drinking water recommended by the Department of National Health and Welfare, Canada (NHW 1969, p. 14).

The usual activated sludge treatment of wastewaters removes 20% to 45% of the cadmium in the wastewaters. Physico-chemical processes are much more efficient. An initial concentration of 5.0 mg/L of cadmium has been reduced to 0.02 mg/L after lime precipitation—activated carbon treatment (Cohen 1977). At a plant in Dallas, Texas, in which the activated sludge treatment was followed by lime precipitation, filtration and passage through activated carbon, the cadmium concentration in the effluent was 0.008 mg/L (cadmium influent concentration, 0.013 mg/L) (Cohen 1977).

It is recommended that the total cadmium concentration in raw public water supplies does not exceed the maximum concentration of 0.01 mg/L permitted in drinking water. For raw waters which will have advanced physico-chemical treatment, the recommended limit for total cadmium concentration is 0.02 mg/L. The recommended limit for cadmium concentration in domestic water supplies in the United States is 0.010 mg/L (EPA 1976, p. 27). The same value was also recommended for Australian domestic water supplies (Hart 1974, p. 49).

Aquatic Life and Wildlife

Bioaccumulation by Aquatic Organisms

Cadmium is taken up in large amounts by aquatic organisms. *Elodea canadensis* (Canadian pondweed), a rooted aquatic macrophyte, took up cadmium from both sediments and water. *Ceratophyllum demersum* (coontail), a non-rooted aquatic, absorbed cadmium from the water. Concentrations of cadmium in the plants reached a maximum in June or July and decreased by early August. The decrease was due to a release of cadmium from the plants (Mayes 1976). Concentration factors for the roots and shoots of *Elodea* were 20 000 and 21 000, respectively (Ravera *et al.* 1973). *Spartina alterniflora* was able to absorb cadmium from marsh sediments and to translocate it to the aerial parts (Valiela *et al.* 1974).

A concentration factor of 6030 was determined for the duckweed *Lemna valdiviana* and factors up to 9500 were reported for the aquatic fern *Salvinia natans* in mon-

oxenic cultures (Cd levels from 0.01 to 0.5 mg/L). The vulnerability of the plants, however, changed when they were in competition with each other. *Lemna valdiviana* became more susceptible and *Salvinia natans*, the more successful competitor, less susceptible to cadmium uptake. As these two plants are part of the aquatic food web, their concentration factors may be important (Hutchinson and Czyrska 1972). Landner and Jernelöv (1969) reported a concentration factor of 100 to 150 in *Lemna minor* roots.

The accumulation of cadmium by *Chlorella pyrenoidosa* grown at pH 7 was twice as great as at pH 8 at concentrations up to 0.5 mg Cd/L. At concentrations beyond this, cadmium itself had an effect on the uptake into the cells (Hart 1975). Soeder *et al.* (1978) also demonstrated an increase in the uptake of cadmium by algal cells at lower pH. Cells of *Coelastrum proboscideum* exposed to 0.01 mg Cd/L at pH 4 took up 67% of the cadmium from the medium within 24 h and at pH 5.8, 60%. When exposed to 0.02 and 0.04 mg/L at pH 4, the percentage decrease of the cadmium concentration in the medium was 71% and 69% and at pH 5.8, 46% and 43%, respectively. Therefore more cadmium was removed from the medium by the cells at the lower pH level.

Caddisflies (Trichoptera) and mayflies (Ephemeroptera) accumulated cadmium from a stream containing 0.0106 mg Cd/L by a factor greater than 300 (Whitton and Say 1975). Mayflies, caddisflies, stoneflies and a species of snail accumulated cadmium up to 30 000 times the concentration of cadmium in the test water (0.003 mg/L); this was 3 times that accumulated by flagfish. The increase of cadmium in stoneflies and snails was (on a mg/kg basis) in proportion to the concentration of cadmium in the water even at the highest levels tested, but the cadmium appeared to reach a plateau in caddisflies exposed to the higher cadmium levels (0.085 and 0.238 mg/L) (Spehar *et al.* 1978). The accumulation of cadmium by largemouth bass and bluegill also appeared to reach an equilibrium between the concentrations of metal in the water and in the tissues. The maximum total body burden by the bass was 8 times (0.008 mg Cd/L in the test water) and 15 times (0.08 mg Cd/L in the test water) greater than the controls. The maximum in the bluegill was 6 times (0.008 mg Cd/L), 20 times (0.08 mg Cd/L) and 210 times (0.85 mg Cd/L) greater than the controls (Cearley and Coleman 1974). Crayfish (*Orconectes propinquus propinquus*), which is used as a food, accumulated cadmium in a fairly short time to levels higher than those considered safe for human consumption. The water in which the crayfish were living contained 0.010 mg Cd/L, which was the level of cadmium given as a guideline for potable water supplies (Gillespie *et al.* 1977). Levels of cadmium in marine copepods (zooplankton) were from 5 to 8 times higher than in the predator Arctic cod (*Boreogadus saida*). Since the copepods formed 20% of the diet of the cod, there did not appear to be biomagnification of the cadmium. The cadmium concentration ranged from 0.35 to 1.6 mg/kg in the liver and from 0.26 to 1.5 mg/kg in the whole fish (dry weight) (Bohn and McElroy 1976).

Concentrations of cadmium in the muscle of fish from Baie du Doré and Toronto Harbour, an unpolluted and a polluted body of water, were 0.06 and 0.13 mg/kg, respectively, a ratio of 1:2. The ratio of cadmium in the liver was 1:3 between the two regions (Brown and Chow 1975). Lucas *et al.* (1970) measured cadmium concentrations from 0.062 to 0.14 mg/kg in whole fish from Lakes Superior, Michigan and Erie. The fish livers appeared to concentrate the cadmium, as concentrations ranged from 0.06 to 1.4 mg/kg with most values around 0.4 mg/kg. Mount and Stephan (1967) studied the distribution of cadmium between the organs of bluegills exposed for periods of 30, 60 and 90 days. There was a substantial accumulation of cadmium in the kidney, liver, gill and gut; less accumulation in the spleen; and no significant accumulation in the muscle and bone. The concentrations in the liver and gill were closely correlated with exposure to cadmium in sublethal concentrations. After acute exposure, the gill concentration was much higher but the liver concentration was very low, and after a 3-day lethal exposure, there was virtually no cadmium in the liver. After exposure to 8 mg Cd/L for a mean of 30 days (range 23-57 days), fish had a concentration of 0.252 mg Cd/kg dry tissue in the gill at death. In water containing 16 mg Cd/L for a mean of 5 days (range 3-7 days), the cadmium concentration in the gill was 0.634 mg/kg (dry tissue).

Very little evidence was found of accumulation through the food web (Fleischer *et al.* 1974, p. 308). Concentrations of cadmium in bottom sediments, worms, clams, omnivorous fish, carnivorous fish, and water were about 2.0, 1.1, 0.6, 0.03, 0.03 mg/kg and 0.0006 mg/L, respectively (Mathis and Cummings 1973). In contrast with this, some authors considered that accumulation of cadmium might occur through the food web (Sanford 1974; Coughtrey and Martin 1976). The biological half-life in trout was 17 to 32 days and in guppy, 18 days, warm water species eliminating cadmium faster than cold water fish. No evidence was found that supported higher concentrations of cadmium in predators than in prey (Smith and Huckabee 1973, p. 300).

Toxicity to Aquatic Organisms

Cadmium has been shown to be extremely toxic to aquatic organisms. A number of studies quoted in the International Joint Commission Water Quality Objectives Subcommittee Report (IJC 1976) showed a great variability between organisms, for example, between the different classes of phytoplankton.

Phytoplankton——Green algae (Chlorophyta) have very different sensitivities to cadmium ions. There was a 70% inhibition of photosynthesis in *Chlorella pyrenoidosa* by 0.1 mg Cd/L, but 10 times the amount of cadmium (1.0 mg Cd/L) had the same effect in *C. vulgaris* (Burnison *et al.* 1975). In another study with different conditions, 0.06 mg Cd/L inhibited the growth of *C. vulgaris* by 50% of the control growth (Rosko and Rachlin 1977). Growth inhibition of *C. pyrenoidosa* was affected by pH. The same concentration of cadmium was more toxic at pH 7 than at pH 8 (Hart and Scaife 1977). The growth of *Scenedesmus quadricauda* (Chlorophyta) was significantly inhibited by 0.006 mg Cd/L (Klass *et al.* 1974). Mitochondria (organelles essential for cell respiration) of *Ankistrodesmus falcatus, Chlorella pyrenoidosa* and *Scenedesmus quadricauda* were studied by electron microscopy after the algae were exposed to low levels of cadmium chloride. Concentrations of 0.03, 0.05 and 0.1 mg/L caused degenerative changes, the severity increasing with an increase in cadmium concentration (Silverberg 1976).

A blue-green alga, *Anacystis nidulans,* was 5 to 10 times more sensitive than *C. pyrenoidosa,* and its growth was inhibited by 0.05 mg Cd/L. This suggested that *C. pyrenoidosa* was less sensitive than stated by Burnison *et al.* (1975). One of the explanations postulated for differences in sensitivity between blue-greens and other algae concerned the absence of a chloroplast in blue-greens. The cadmium ion has been shown to affect at least two of the photosynthetic groups of enzymes containing thiol, the CO_2 fixation stage more so than the oxygen evolution stage. The site of photosynthesis in blue-greens is the cytoplasm, while in green algae it occurs in the chloroplasts. It is possible that a large amount of the cadmium entering the cell never reaches the chloroplast but is bound to metallothionein in the cytoplasm (Hart 1975). This, however, would not explain the greater sensitivity of the green alga *S. quadricauda.* Cadmium competes with iron and manganese, both of which are involved in photosynthesis (Hart and Scaife 1977).

Macrophytes——Aquatic macrophytes were also affected by cadmium. A concentration of 0.007 mg Cd/L caused reduced chlorophyll levels, turgor, and stolon development in the bushy pondweed *Najas guadalupensis* (Cearley and Coleman 1973).

Zooplankton——As with phytoplankton, the toxicity of cadmium ions to zooplankton varied according to the species tested. Acute toxicities, 48-h LC_{50}'s, ranged from 0.065 mg Cd/L for *Daphnia magna* (Biesinger and Christensen 1972) and 0.055 mg Cd/L for *Daphnia hyalina* to 3.8 mg Cd/L for *Cyclops abyssorum prealpinus* (Baudouin and Scoppa 1974). Reproduction of *Daphnia magna* had a 16% impairment by 0.00017 mg Cd/L in water of hardness 45 mg/L as $CaCO_3$ (Biesinger and Christensen 1972).

Cadmium stress reduced the numbers and biomass in populations of *Daphnia galeata mendotae* and increased the population variability, the turnover rate and the proportion of ovigerous females. In populations where extinction did not occur, the birth rates increased sufficiently to match the death rates (as can also occur with other environmental stresses) through an increased mean number in the brood, although the average size of the individual eggs decreased. This indicated that the population sensitivity was not as great as that of an individual. The increase in brood number was mainly due to the increased food supply per individual because of the reduced standing crop (biomass and numbers). An increase in postnatal death was significant at 0.002 mg Cd/L. Prenatal mortality was the most sensitive indicator of cadmium stress; 0.001 mg Cd/L caused a 50% reduction in survival compared with that of the control embryos. The population numbers were reduced to 50% of the controls by 0.0077 mg/L (Marshall 1978).

Philodina acuticornis (Rotifera) was similar in sensitivity to some of the microcrustaceans. The 96-h EC_{50} (the estimated concentration of cadmium that would affect 50% of the rotifers in 96 h) was 0.5 mg/L for cadmium chloride and 0.2 mg/L for cadmium sulphate (water hardness 17 mg/L as $CaCO_3$). Cadmium was the most toxic of a number of metals tested including Hg, Cu and Zn. In water of hardness 53 mg/L (as $CaCO_3$), the 96-h EC_{50} for cadmium sulphate was 0.3 mg/L (Buikema *et al.* 1974).

Insects——Aquatic insects were less sensitive than zooplankton. The 96-h LC_{50}'s ranged above 1.2 mg/L, for example, 2.0 mg/L for caddisflies compared with 0.1 mg/L for *Daphnia* (Warnick and Bell 1969); 3.4 mg/L for caddisflies and 1.2 mg/L for chironomids (Rehwoldt *et al.* 1973); and 18.0 and 28.0 mg/L for *Pteronarcella badia* (stonefly) and *Ephemerella grandis grandis* (mayfly), respectively (Clubb *et al.* 1975b). Amphipods (*Gammarus* 0.07 mg Cd/L) were similar in sensitivity to zooplankton (Rehwoldt *et al.* 1973).

Spehar *et al.* (1978) studied the toxicity of cadmium to four species of insects (*Pteronarcys dorsata, Hydropsyche betteni, Brachycentrus* sp. and *Ephemerella* sp.) and one snail (*Physa integra*) over a period from 4 to 28 days. The 28-day LC_{50} value for snails was 0.0104 mg/L, approximately 11 times lower than the 7-day LC_{50}; for mayflies at <0.003 mg/L, the mortality was greater than 50% with the lowest concentration tested, but mortality was only significant in the last week of exposure in all concentrations tested (0.003 to 0.238 mg/L). Caddisflies and stoneflies were less sensitive and no significant decreases in survival occurred after 28 days in the test concentrations. The

behaviour of caddisflies at 0.0855 and 0.238 mg/L was different from the controls, and the larvae were usually out of their cases, free-swimming and curling vigorously. This behaviour was similar to that observed in fish exposed to cadmium (Cearley and Coleman 1974; Benoit *et al.* 1976; Spehar 1976). The lowest effect concentrations of cadmium to the invertebrates after 28 days of exposure were similar to those for fish over their complete life cycle in water of similar quality. Early developmental stages of the invertebrates were not studied by Spehar *et al.* (1978) but they have been shown to be more sensitive to cadmium toxicity by other authors (Clubb *et al.* 1975b) as has been shown for juvenile fish (Benoit *et al.* 1976; Spehar 1976).

Wentsel *et al.* (1977) demonstrated that cadmium contamination of sediments caused developmental retardation of *Chironomus tentans* (midge) larvae. There was a significant difference between the survival time of larvae in the control sediment (0.6 ±0.4 mg Cd/kg dry weight; 6.13% organic matter) and in heavily contaminated sediment (1030 ±5 mg Cd/kg dry weight; 8.08% organic matter). The length of the larvae differed from a mean of 1.83 cm in the control sediment to 0.82 cm in sediment containing 960 ±9 mg Cd/kg and the weight differed from 2.86 mg in the control sediment to 0.2 mg in the contaminated sediment. No significant correlation was found between the percentage of organic matter or texture of the sediment and the growth of the midge larvae. In a study of Palestine Lake, Indiana, larvae were not found in the highly contaminated parts, but were found in areas of moderate or little contamination. Their growth was probably retarded in the former areas. The oligochaete *Limnodrilus* sp. was dominant in the heavily contaminated parts of the lake (Wentsel and McIntosh 1977).

Snails———Immature snails (*Physa gyrina* Say) were shown to be 3 times as sensitive to cadmium (96-h LC_{50}, 0.41 mg/L) as mature snails (96-h LC_{50}, 1.37 mg/L). This agreed with experiments on fish which indicated the early life stages were the most sensitive. Accumulation of cadmium over a 24-h period (0.55 mg/L·h) exceeded the elimination rate (0.229 mg/L·h) over the next 24 h, thus leading to accumulation of cadmium in the snail (Wier and Walter 1976).

Fish———There was also variation in the sensitivity of different fish species to cadmium, the salmonids apparently being the most sensitive. Acute toxicity varied according to time of exposure and species tested. The 96-h LC_{50} in water with a hardness of 200 mg/L as $CaCO_3$ was 7.2 mg/L (mean of five tests) for fathead minnows, while the 8-day LC_{50}, indicating a cumulative mortality, was 0.45 mg/L (Pickering and Gast 1972). The 7-day LC_{50} for rainbow trout was 0.008 to 0.01 mg/L cadmium (Ball 1967), and the 10-day LC_{50} was similar at 0.005 to 0.007 mg/L, trout being much more sensitive than some of the other species of fish (Kumada *et al.* 1972). The average mortality for coho salmon (*Oncorhynchus kisutch)* exposed to 0.008 and 0.012 mg Cd/L for 6 days was 14.3% and 53.1%, respectively. In the same study, which also concerned the toxicity of copper, different responses to copper were reported for the two sets of fish. Copper elicited a rise in cortisol levels, which is a response to stress, but the reaction to cadmium was different, as the control and cadmium treated fish had similar increases in cortisol owing to handling stress. Cadmium exposure (0.0045 mg/L) did not prevent the fish from adapting to sea water (Schreck and Lorz 1978).

The difference in sensitivity between fish species was also demonstrated by longer term studies of 30, 60 and 90 days, using embryo-larval and early juvenile stages. The most sensitive species investigated was the brook trout in soft water (45 mg/L as $CaCO_3$), with a maximum acceptable toxicant concentration (MATC) of 0.0011 to 0.0038 mg Cd/L at 10°C (Eaton *et al.* 1978) and 0.0017 to 0.0034 mg Cd/L at 9°C (Benoit *et al.* 1976) over 90 days. Lake trout, coho salmon and brown trout tolerated MATCs of 0.0044 to 0.0123, 0.0041 to 0.0125, and 0.0038 to 0.0117 mg Cd/L, respectively, for 60 days at 10°C in soft water. In a 30-day study under similar conditions, northern pike, smallmouth bass and white sucker withstood MATCs of 0.004 to 0.0129, 0.0043 to 0.0127, and 0.0042 to 0.012 mg Cd/L at 16°C, 20°C and 18°C, respectively (Eaton *et al.* 1978). Sauter *et al.* (1976) studied brook trout at 10°C in water of hardness 36 mg/L as $CaCO_3$; it tolerated a MATC of 0.001 to 0.003 mg Cd/L in 60 days, which is similar to that found by Eaton *et al.* (1978) and Benoit *et al.* (1976). The MATC for channel catfish at 22°C in the same water was 0.011 to 0.017 mg Cd/L. In harder water, 187 mg/L as $CaCO_3$, the MATC for brook trout was 0.007 to 0.012 mg Cd/L over 60 days, but the toxicity to catfish was little affected by hardness, as the MATC was 0.012 to 0.017 mg Cd/L (Sauter *et al.* 1977). The MATCs over 60 days for the fathead minnow and bluegill in water with a hardness of 200 mg/L as $CaCO_3$ were 0.037 to 0.057 mg/L at 24°C (Pickering and Gast 1972) and 0.031 to 0.080 mg/L at 26°C (Eaton 1974).

Factors Affecting Cadmium Toxicity

Water hardness affected the toxicity of cadmium and there was also variation in sensitivity to cadmium between species. The 96-h LC_{50} for fathead minnows ranged from 0.63 to 1.05 mg Cd/L in soft water (20 mg/L as $CaCO_3$). The difference between species is illustrated by the green sunfish and the minnow. The latter was more sensitive in soft water; the 96-h LC_{50} was between 0.63 and 1.05 mg Cd/L for the minnow compared with 2.84 mg Cd/L for the sunfish (hardness 20 mg/L as $CaCO_3$). In hard water (360 mg/L as $CaCO_3$), the 96-h LC_{50} was 72.6 to 73.5 mg Cd/L for the minnow and 66 mg Cd/L for the sunfish (Pickering and Henderson 1966).

The solubility of cadmium in hard and soft water was calculated to be about the same, but the toxicity of cadmium to rainbow trout decreased in hard water. The level of no effect was from 0.014 to 0.021 mg/L at a hardness of 315 mg/L as $CaCO_3$ compared with 0.0007 to 0.0015 mg/L at a hardness of 31 mg/L as $CaCO_3$. The differences were thought to be due to an antagonistic effect of calcium (Davies 1976) and maybe magnesium (Hutchinson and Czyrska 1972).

Water hardness also affected the uptake of cadmium by aquatic animals and plants. The rate of uptake was initially faster in hard water. The total amount taken up by the tissues of *Nitella flexilis* (alga), *Elodea canadensis*, snails, catfish and guppies was greater in soft than in hard water (150 mg/L as $CaCO_3$) (Kinkade and Erdman 1975).

The effect of the chelating capacity of natural waters on cadmium toxicity was investigated in 22 lakes. Where the chelating capacity was higher than 2 µmol/L of copper equivalent, the toxic effect of cadmium on *Selenastrum capricornutum* growth was completely or strongly reduced (Chiaudani and Vighi 1978).

Rehwoldt *et al.* (1972) stated that a rise in temperature from 15°C to 28°C did not affect the toxicity of cadmium to several species of freshwater fish, but Eisler (1971) found a 2- to 3-fold increase in the 96-h TL_m for cadmium with *Fundulus heteroclitus* in sea water when the temperature was raised from 5°C to 20°C.

The sensitivity to cadmium of the mummichog *Fundulus heteroclitus* L. in fresh water was significantly greater at lower levels of dissolved oxygen. The 96-h LC_{50} ranged from 1.3 to about 3.0 mg Cd/L at 2.3 and 8.5 mg/L dissolved oxygen, respectively. Different oxygen levels in sea water did not have the same effect on cadmium toxicity as in fresh water, since the initial uptake of cadmium is via the gastrointestinal tract. Water was taken in through the gills to a much greater extent by the mummichog when it was in fresh water and more water would pass over the gills when oxygen was low (Voyer *et al.* 1975). A different effect of dissolved oxygen on cadmium toxicity was observed with insects. An increase in the concentration of dissolved oxygen increased the metabolic rate and also cadmium uptake by the mayfly, stonefly and caddisfly species tested. The data suggest that cadmium may have less effect on aquatic insects in an oxygen deprived environment than in one with normal oxygen concentrations (Clubb *et al.* 1975a).

Sublethal Studies

Sublethal studies which exposed early life stages of fish indicated that a 60-day exposure period was appropriate for determining larval sensitivity, as brook trout sensitivity to cadmium was greater than after a 30-day exposure period. Growth was reduced at a cadmium concentration of 0.0038 mg/L in water with hardness of 45 mg/L as $CaCO_3$ (Eaton *et al.* 1978). Bluegill were shown to survive and spawn successfully when exposed to a higher concentration (0.031 mg Cd/L) in water with a hardness of 200 mg/L as $CaCO_3$ (Eaton 1974). There were no adverse effects on the survival, growth or reproduction of fathead minnows at cadmium concentrations between 0.0045 and 0.037 mg/L at a water hardness of 200 mg/L as $CaCO_3$ (Pickering and Gast 1972). Acclimation to cadmium during exposure in the egg stage was demonstrated for rainbow trout fry. Eggs were exposed for one month in soft water (27 mg/L as $CaCO_3$), and the "no-effect" concentration of cadmium for fry hatching from cadmium-exposed eggs was between 0.0034 and 0.0071 mg Cd/L and for fry not exposed as eggs, between 0.0007 and 0.0015 mg Cd/L, indicating that the fry had become acclimated and more resistant to the effects of cadmium (Davies and Goettl 1976).

Sauter *et al.* (1976) estimated that the MATC for brook trout (*Salvelinus fontinalis*) in soft water (37 mg/L as $CaCO_3$) was between 0.001 and 0.003 mg/L cadmium and in hard water (188 mg/L as $CaCO_3$), between 0.007 and 0.012 mg/L cadmium. The MATC of $\geq$0.017 mg Cd/L for channel catfish was based on the percentage survival after 30 days in soft water and the effects on total length and wet weight after 60 days of exposure in hard water. Due to difficulties in feeding, no figure was suggested for walleye.

The mean safe exposure levels of cadmium for the flagfish *Jordanella floridae* were given as 0.0074, 0.003 and 0.0034 mg/L and compared well with 0.0041 mg/L from previous tests in soft water (water hardness of approximately 44 mg/L as $CaCO_3$) (ERLD 1976).

An application factor for cadmium (MATC/96-h LC_{50}) for flagfish (*Jordanella floridae*) was calculated to be between 0.0016 and 0.0032, based on the MATC, which was between 0.004 and 0.008 mg Cd/L, and the 96-h LC_{50} of 2.5 mg Cd/L. A similar factor was obtained from experiments with hard water. Abnormal behaviour (uncontrolled swimming, loss of equilibrium, etc., attributed to the inhibition of acetylcholinesterase) was reported for a number of fish species, especially male fish, when exposed to levels of cadmium which caused death and inhibited reproduction (Spehar 1976).

Benoit *et al.* (1976) determined a MATC of between 0.0017 and 0.0034 mg/L cadmium for brook trout (*Salvelinus fontinalis*) in Lake Superior water (hardness 44 mg/L as $CaCO_3$) based on three generations of fish. The 96-h LC_{50} was indeterminate, and the authors made no

attempt to calculate an application factor. Other fish species have also given indeterminate acute toxicities.

Three to four year old male trout (*Salvelinus fontinalis*) were used in studies to determine whether sublethal levels of cadmium affected reproductive physiology and thus reproductive success. Hemorrhagic necrosis occurred in the testes of fish exposed to 0.025 mg/L cadmium for 24 h followed by 6 days in uncontaminated flowing water. Similar but less severe lesions were present when fish were exposed to 0.010 mg/L for 21 days. There was no effect on secondary sexual characteristics or damage to the primordial germ cells, the initial sites of spermatogenesis, in either set of fish, but in cadmium-treated fish, the period of functional maturity was terminated 2 weeks earlier than in the controls. The testicular changes caused by cadmium were only observed in mature or nearly mature fish (Sangalang and O'Halloran 1972). Exposure of 0.001 mg/L cadmium for at least three months prior to the onset of sexual maturation had a direct effect on steroid enzyme systems (androgens are concerned in the maintenance of spermatogenesis). Both testosterone and 11-ketotestosterone levels in the plasma were higher than in the controls, suggesting that cadmium has an adverse effect on the utilization and clearance of the steroids (Sangalang and Freeman 1974).

Sanford (1974) also reported the occurrence of hemorrhagic testicular necrosis in brook trout exposed to water containing 0.01 and 0.025 mg Cd/L for 24 h. Under natural environmental conditions, if cadmium is present for only a short time at a critical period in the life cycle, breeding may be affected. Pathological changes have also been found in the gills, intestines and kidney of brook trout (Gardner and Yevitch 1970).

Osmoregulation was affected by sublethal levels of cadmium. Both the plasma and tissue electrolyte levels and the tissue water content were affected, but it could not be determined whether these changes were significant to the fish (goldfish) in its natural environment (McCarty and Houston 1976). Cadmium (0.1 mg/L) was shown to increase the excretion of calcium, which is essential in a number of physiological functions, such as the permeability of cell membranes to water and ions, and the maintenance and development of membrane potential in muscles and nerves. Calcium is also a vital component of the skeleton. Even though no visible symptoms of toxic reaction were present initially after an exposure of 9 to 10 days, some physiological functions had been affected, as the fish were excitable and had occasional uncoordinated swimming movements. Cadmium was also shown to accumulate in the Islets of Langerhans in the pancreas, affecting blood sugar levels and thus carbohydrate metabolism. It was suggested that these sublethal effects might be more serious than more obvious ones, such as fish kills, since whole populations might be less successful in the natural environment and this could go unnoticed (Larsson *et al.* 1976).

Cadmium has been shown to affect the behaviour of fish. The stone loach *Noemacheilus barbatulus* exposed to 3.4 mg Cd/L (34-day LC_{50}; 7-day LC_{50} = 4.5 mg Cd/L) did not hide during the daylight hours as it did in control tanks and in the wild, and the authors proposed that as a result, the presence of cadmium might have made the fish more susceptible to predators (Solbé and Flook 1975).

Fathead minnows (*Pimephales promelas*), prey of largemouth bass, were more vulnerable after exposure to 0.025 mg Cd/L (water hardness 349 mg/L as $CaCO_3$) for 21 days, a level below the MATC of between 0.037 and 0.057 mg Cd/L in water of hardness 200 mg/L as $CaCO_3$ that was estimated by Pickering and Gast (1972). Acute exposure to high levels of cadmium (0.50 and 0.375 mg/L in water of hardness 41 mg/L as $CaCO_3$) had a similar effect. The fish had altered behaviour patterns which included abnormal schooling behaviour (Sullivan *et al.* 1978).

The locomotor activity of the bluegill *Lepomis macrochirus* was increased 1.5 and 7.8 times on exposure to two of the test concentrations of cadmium, 0.1 and 0.25 mg Cd/L, respectively. The increase in activity did show a concentration-dependent relationship at these concentration values (Ellgaard *et al.* 1978). Exposure to 0.5 mg/L decreased the activity to 0.6 of the controls, but this concentration caused 30% mortality after 2 weeks exposure (water hardness 105 mg/L as $CaCO_3$). It must be noted that the fish had been moved to a limited space immediately prior to these series of experiments and this may also have had an effect on their activity.

Cadmium caused teratogenic effects in trout embryos; 28% had abnormalities after exposure to 0.5 mg Cd/L, 9% after exposure to 0.05 mg Cd/L, and 7% after exposure to 0.005 mg Cd/L (Birge *et al.* 1974). Teratogenicity in fish was demonstrated in a series of experiments by Weis and Weis (1977). *Fundulus heteroclitus* (killifish) eggs were dosed with cadmium ranging from 0.01 to 10 mg/L. Axis formation was affected in early stages of development by 0.01, 0.1 and 1.0 mg/L in 23% to 40% of the eggs. Retarded development occurred in 32% of the embryos exposed to 10 mg Cd/L compared with 4% in the controls. The occurrence of defective development was less frequent in embryos exposed at a late stage of development. Since in the field exposure would occur at all stages in the life cycle, embryonic defects could be more severe than indicated in these experiments. Eaton (1974) reported severe physical abnormalities in bluegills, including pericardial and abdom-

inal edema, lordosis and deformed dorsal fins on exposure to 0.239 mg Cd/L.

The Interactions of Cadmium with Other Elements and Compounds

Hutchinson and Czyrska (1972) studied the effects of cadmium and zinc ions, alone and together on two floating aquatic plants, duckweed (*Lemna valdiviana*) and a fern (*Salvinia natans*). In monoxenic cultures, there was slight inhibition of growth in *Lemna* by 0.1 mg Cd/L, the effects becoming more marked with time. The suppression of growth by this concentration was more severe in *Salvinia.* When the two plants were grown together the effect of cadmium on *Lemna* was enhanced, but the effect was less on *Salvinia,* which was the more successful of the two in competition in the control series. Zinc stimulated the growth of both species when they were grown alone, yet when both cadmium and zinc were present, the growth stimulation caused by zinc was completely suppressed by the cadmium. When they were grown in competition with each other in the presence of a mixture of zinc and cadmium, the toxicity of cadmium was accentuated. The presence of zinc enhanced the inhibitory effect of cadmium on the growth of *Lemna,* and to a lesser extent on *Salvinia,* the species less affected by the stress of competition.

Selenium has also been shown to have a protective effect against cadmium toxicity with respect to testicular necrosis, teratogenesis, and a reduction in mortality rate (Smith 1973, p. 276; Parizek *et al.* 1974; Friberg 1976). Selenium ameliorated the toxic effects of cadmium, and inhibition of growth was much less than the inhibition caused by either cadmium or selenium when these two elements were alone. This is an example of antagonism by the two elements, as *Chlorella* growth was reduced to 55% of the controls by 0.05 mg/L cadmium and to 70% by 0.05 mg/L selenium when they were alone. In combination, the growth was 81% of the control. At 0.05 mg/L cadmium and 0.1 mg/L selenium, growth was stimulated compared with the control (Hutchinson 1973; Hutchinson and Stokes 1975). The mechanism of selenium detoxification of metals is not fully understood. The affinity of Cd^{2+} for sulphydryl groups is very strong, but the affinity for the Se analog (R-Se-H) is much greater. In addition, selenium can bind to sulphydryl groups to form R-S-Se-H derivatives. Although selenium can counteract the toxicity of cadmium, it also causes an increase in the retention of the metal and alters the tissue distribution of cadmium. The effects of selenium on cadmium depend on the route of entry into the body and the relative doses. The reverse, i.e., cadmium protection against the toxicity of selenium, has also been observed (Rimerman *et al.* 1977).

Chlorella sp. (Chlorophyta) accumulated up to 500 times the concentration of cadmium that was present in solution and suggests that cadmium may be transported against a diffusion gradient. The light-dependent nature of cadmium accumulation also provided support for this being an active process requiring adenosine triphosphate (ATP) derived from photophosphorylation. Low temperatures greatly reduced its uptake, indicating an enzymatic process. *Chlorella* cultures exposed to 0.5 mg Cd/L accumulated twice as much cadmium at pH 7 as at pH 8. The complex of iron-EDTA in the culture medium blocked the accumulation of cadmium by *C. pyrenoidosa* and relieved the growth inhibition caused by cadmium. The exact mechanism of this blocking and that by manganese is not yet fully understood. Once cadmium enters the cell it is tightly bound to subcellular components (Hart 1975). The uptake of cadmium by diatoms varied with the growth phase, the maximum occurring at the end of the phase. The amount taken up correlated with the concentration of cadmium in solution. In water containing EDTA, cadmium competed with it and there was little uptake by the cells (Cossa 1975).

Lead affected the toxicity of cadmium in two ways, depending on the ratio of the two metals. Synergism was indicated in solutions where the concentration of cadmium was greater than that of lead, as the reduction of primary production was more severe than would be expected when cadmium was alone at the same concentration. When the concentration of lead was greater than that of cadmium, there was an antagonistic effect (Pietilainen 1976).

Fathead minnows were used to test the toxicity of a mixture of cadmium, copper and zinc in concentrations ranging from "toxic" to "safe." The experimental period was 12.5 months, and water hardness was 200 mg/L as $CaCO_3$. There was no indication of a strict summation of toxicities. A lethal threshold was attained in the mixture when each metal was present at a concentration of 0.4 or less of its individual lethal threshold. Therefore, the toxicity of the trimetal mixture could not have been predicted closely from results of tests with individual metals. Cadmium, when alone, reduced the number of eggs that hatched, but this specific toxic action seemed to be reduced in the trimetal mixture, as the hatch rate (93%) was better than in the controls (79%). The chronic toxicity of the trimetal mixture, with metals in proportion to individual concentrations shown to be sublethal, was more toxic than would have been predicted from strict summation of the individual toxicities (Eaton 1973).

In contrast, when cadmium was tested in combination with malathion, methoxychlor, and methoxychlor-Aroclor 1254, the toxicant combinations caused mortalities equal to the arithmetic sums of the individual toxicities (Bahner and Nimmo 1975). These organic compounds were tested under estuarine conditions using pink shrimp (*Penaeus duorarum*). There may be different mechanisms causing toxicity in salt and fresh water, especially in fish, due to differing osmoregulation (Cairns *et al.* 1975).

Sediments

The role of cadmium-contaminated sediments in toxicity to fish and amphibian eggs and fry was studied using rainbow trout (*Salmo gairdneri*), goldfish (*Carassius auratus*) and narrow-mouthed toad (*Gastrophryne carolinensis*). Sediment was collected from a stream carrying a diverse community of aquatic biota, and it was used in its natural condition or spiked with four different concentrations of cadmium in synthetic water with a hardness of 200 mg/L as $CaCO_3$ and pH 7.9 at 22°C. The embryonic mortality was appreciably higher over cadmium-spiked sediments than for the controls and the frequency of teratogenic abnormalities was also higher. Only small amounts of sediment-bound cadmium were released to the overlying water and 95.6% of the cadmium was retained in the sediments after 20 days. The sediment appeared to be more lethal to the eggs than to the free living larvae and fry, as the percentage mortality after hatching was less than during embryonic development. Ten days after hatching, the percentage survival over cadmium-spiked sediment was 80%, 53%, 37% and 11% for sediment spiked with 0.1, 1.0, 10.0 and 100.0 mg Cd/kg, giving actual levels of 1.40, 2.0, 12.7 and 113.0 mg Cd/kg in the sediment and levels of 0.0047, 0.0068, 0.0075 and 0.0072 mg Cd/L in the water. The control survival was 94%. Goldfish and toads were not as sensitive as trout to the highest concentrations of cadmium. Contaminated sediments warrant serious consideration in areas where contamination exists or could occur, as bottom dwelling organisms or eggs could be more seriously affected than expected from results of water related bioassays (Birge *et al.* 1977).

Recommended Objective for Aquatic Organisms

Aquatic organisms have been shown to be very sensitive to cadmium. Levels ranged from 0.0015 mg/L, which caused no significant effect in the more sensitive species of fish, to 0.00017 mg/L, which caused a 16% impairment of reproduction in *Daphnia magna* (water hardness 45 mg/L as $CaCO_3$). Levels of cadmium in hard water producing the same effect were higher (up to 0.037 mg/L), probably due to calcium, which is thought to have an antagonistic effect on cadmium toxicity (Davies 1976).

Combinations of metals have been shown to have a synergistic effect, lowering the lethal threshold to at least 0.4 of its value for the single metals in acute toxicity studies (Eaton 1973).

Cadmium affects vital physiological functions and has been shown to be teratogenic.

Opinions on the possibility of accumulation of cadmium through the food web differ. Organisms have been shown to accumulate cadmium directly from water, with factors ranging from 100 to 9000. This presents the possibility of bioaccumulation, although some studies analyzing the cadmium content of various members of the food web have discounted biomagnification.

Considering these facts and that cadmium will not be the sole pollutant in a natural body of water, the recommended permissible level for aquatic life is 0.0002 mg/L. In the very soft waters found in some parts of Canada, e.g., 12 mg/L as $CaCO_3$ in northwestern Ontario, this level may be too high and should be reduced, especially when taking into account the effects on zooplankton.

Due to the difficulty experienced by a number of researchers in attempting to obtain reliable acute toxicity data (Sauter *et al.* 1976), the recommended level may change by quite a large factor when further research has been done.

Hard water decreased the toxicity of cadmium to aquatic organisms, and considering one of the studies where both types of water were used (Pickering and Henderson 1966), a recommended limit of 0.004 mg Cd/L could be possible at a hardness of >360 mg/L as $CaCO_3$ (the hardness used by Pickering and Henderson). A moderately hard water of 200 mg/L as $CaCO_3$ may have the same effect, but the figure of 0.004 mg Cd/L cannot be used until more studies have been done.

Wildlife

Samples of plants and muskrat tissues from areas which were variously affected by heavy metal pollution were analyzed for cadmium content. The concentrations of cadmium in food plants (10.46 mg/kg) and muskrat liver (0.316 mg/kg) and kidney (1.071 mg/kg) were positively correlated. The muskrats did not appear to be adversely affected, as parameters indicating the physical condition of the animals were not statistically significant when compared with those of muskrats caught in less affected areas (Everett and Anthony 1976).

Insufficient information was found in the literature on which to base an objective for cadmium in natural

waters. The objective recommended for aquatic life, 0.0002 mg/L cadmium, should also be harmless to wildlife.

It may prove possible to recommend the level of 0.02 mg/L total cadmium proposed for watering livestock for wildlife (see next section), but at present there is not enough information to determine whether or not this is feasible.

Water for Livestock

Cadmium in the Animal Body

The reported values on the retention of ingested cadmium by farm animals vary over a wide range from 2% to 18% (Willoughby 1978). Of a single oral dose of Cd(II) only 0.75% was retained by cows (Neathery *et al.* 1974) and 0.35%, by goats (Miller *et al.* 1969) after two weeks. Many elements (Zn, Cu, Fe, Hg, Se, Ca, Co) and compounds (protein, chelating agents) interact with cadmium to modify its rate of absorption (Willoughby 1978).

Lambs fed diets containing cadmium over a six-month period showed metal accumulation in kidney and liver tissues proportional to diet concentrations. Very low concentrations were found in muscles and fat. With diets containing 5 mg/kg cadmium and an average daily intake of 10.8 mg cadmium over a 163-day period, the kidney had a concentration of approximately 58.9 mg/kg (dry weight) and the liver, 14.9 mg/kg (dry weight). Due to different organ weights the total amount of cadmium that accumulated in the liver was greater than that accumulated in the kidney: 4.75 and 2.08 mg, respectively. At the highest diet concentration tested (60 mg/kg or approximately 111 mg/day), the concentrations in the kidney and liver were 769 and 276 mg/kg (dry weight), respectively. The whole-body growth rate decreased progressively with the increase in cadmium intake, starting with a diet containing 15 mg Cd(II)/kg and at an average daily intake of 29 mg Cd(II) (Doyle *et al.* 1974). Accumulations of cadmium in liver and kidney proportional to diet concentrations and reduced growth were also observed in swine. A diet containing 50 mg/kg cadmium and a daily intake of approximately 49 mg resulted in concentrations of 41 mg/kg in kidney and 4.9 mg/kg in liver (Cousins *et al.* 1973). When a single oral dose of Cd(II) was administered to lactating cows, the kidney showed the highest concentration. Based on the total organ content, however, the gastrointestinal tract (tissue and contents) contained 34% of the retained amount, the liver had 32%, and the kidney had 10% (Neathery *et al.* 1974). In goats, the kidney and liver contained 73% (Miller *et al.* 1969).

The kidney appears to be the critical organ for long-term exposure to cadmium. Cadmium also concentrated in the liver but was removed more quickly from this organ than from the kidney. Feces and bile are the main routes for excretion of cadmium from the body (Friberg 1976; Willoughby 1978).

The Toxicity of Cadmium to Farm Animals

The main clinical signs of cadmium toxicity in animals are similar to those of zinc deficiency, namely anemia, retarded gonadal development or degeneration, enlarged joints, scaly skin, liver and kidney damage, and reduced growth (Willoughby 1978).

Dugdale (1978) quoted the following minimum toxic daily intakes of cadmium for various farm animals: horses, 1.7 mg; cattle, 6 to 8 mg; and pregnant ewes, 1 mg/kg of body weight.

Growth, feed intake and water consumption were lower in calves on a diet containing 160 mg Cd/kg (Powell *et al.* 1964). A diet containing 20 mg Cd/kg depressed the growth of turkeys (Supplee 1961).

A high level of dietary cadmium, 3.0 g/day, reduced the milk production of lactating cows to 46% of that during the pretreatment period. With the exception of an increase in the fat content of the milk in the first week of treatment, no change was noticed in the concentrations of milk constituents (fat, non-fat solids, zinc and cadmium) of cadmium-fed cows compared with the control cows (Miller *et al.* 1967).

Cows in cadmium-contaminated areas of Sweden had renal levels of cadmium of 1 to 2 mg/kg (Friberg *et al.* 1974, p. 28). An extensive survey of beef meat, liver and kidney in an agricultural area of West Germany (Kreuzer *et al.* 1976) showed values of <0.005 mg/kg cadmium in the meat, <0.005 to 1.65 mg/kg (median 0.05 mg/kg) in the liver, and 0.008 to 1.95 mg/kg (median 0.229 mg/kg) in the kidney. Very little or no cadmium appeared in the milk of lactating cows (Neathery *et al.* 1974).

Recommended Objective for Water for Livestock

Considering the toxic effects of cadmium on farm animals and its accumulation in kidney and liver, which are consumed by humans, an objective of 0.02 mg/L total cadmium is recommended for livestock water supplies.

Water for Irrigation

Toxicity of Cadmium to Plants

Cadmium above certain levels affected several plant processes such as CO_2 fixation, transpiration-gas stomatal

function, mitochondrial respiration and coupling in addition to interfering with normal metabolism of zinc and iron (Chaney 1978).

Sunflower plants grown in solutions containing 100 or 200 mg Cd(II)/L showed a decrease in net photosynthesis. A 50% decrease was reported at an accumulation of 96 mg/kg of cadmium in leaf tissue (Bazzaz *et al.* 1974). The yield of spinach, soybean and curlycress crops decreased significantly —25% —at concentrations of 4 to 8 mg Cd/kg of soil treated with cadmium-enriched sewage sludge. The lettuce, corn and carrot crops showed a 25% decrease in their yield at applied cadmium concentrations of 13 to 20 mg/kg of soil. Other crops were similarly affected at concentrations of 20 to 50 mg/kg (turnip, field bean, and wheat), 96 mg/kg (radish), and 160 to 170 mg/kg (tomato, zucchini squash and cabbage). Rice was not affected at the highest concentration used in the experiment, 640 mg Cd/kg of soil. Rice grains of plants grown in soil containing 640 mg Cd(II)/kg contained 2 mg Cd(II)/kg. Leafy plants, such as lettuce and spinach, accumulated 70 and 75 mg Cd(II)/kg in the edible part at soil treatments of 13 and 4 mg Cd(II)/kg, respectively. Fruit and seed tissues accumulated much less; wheat had 11.5 mg Cd(II)/kg in the edible part (soil treated with 50 mg/kg) and tomato, 7 mg/kg (soil treated with 160 mg/kg), etc. (Bingham *et al.* 1975). Similarly, Chaney (1978) found that leafy vegetables such as lettuce, spinach, chard, curlycress, turnip greens and beet greens contained higher cadmium concentrations than rice, carrots, wheat grain, corn grain, soybean, radish and red beet. When sludge was applied to give a cadmium concentration in soil of approximately 1.1 mg/kg, the edible parts that showed the greatest accumulation of cadmium were: lettuce, 2.67 mg/kg; carrots, 1.15 mg/kg; and tomatoes, 0.33 mg/kg (Dowdy and Larson 1975).

Visual symptoms of cadmium toxicity to wheat and soybeans began to appear at 2.5 mg cadmium per kilogram of soil (Haghiri 1973), and soybean shoots showed a significant accumulation of cadmium at 1.0 mg/kg of soil (Miller *et al.* 1976).

The uptake of cadmium by plants varied over a wide range, depending on the type of plant, type of soil, acidity of the soil, cation-exchange capacity (CEC), organic matter, and zinc content in the soil. The uptake, for example, was greater in sandy soils than in soils high in clay or organic matter, with low pH or with low CEC (Friberg *et al.* 1974, p. 16; Miller *et al.* 1976). Soil pH, probably more than any other single factor, controls plant uptake of cadmium (Chaney 1978). The uptake of cadmium by plants increased with the increased acidity of the soil (Friberg *et al.* 1974, p. 16).

Less of the chlorophyll green colour was visible, and the dry weight of both tops and roots was reduced in lettuce grown in a nutrient solution containing 2 mg Cd(II)/L (John and Van Laerhoven 1976).

Page *et al.* (1972) have shown that the leafy parts of various plants grown in nutrient solutions containing cadmium showed accumulation and detrimental effects at low concentrations. Plants grown in a solution with a concentration of 0.1 mg Cd/L contained 9.0 mg Cd/kg of bean leaves, 59 mg Cd/kg of tomato leaves, 90 mg Cd/kg of corn leaves, 160 mg Cd/kg of turnip leaves and 280 mg Cd/kg of beet leaves. It is not clear how the cadmium concentration in the nutrient solution relates to cadmium concentrations in soil and there are no data available on the relationship between the cadmium concentration in the leafy tissues and that in the edible parts of various plants. Nevertheless, it is remarkable how much cadmium accumulated in the leaves (Page *et al.* 1972; Turner 1973). Some of the plants—red beets, field bean and turnips—showed a 50% growth reduction and visual symptoms of deterioration at a cadmium solution concentration of 0.2 mg/L.

The mushroom *Agaricus edulis* grown on soil containing 0.16 mg Cd(II)/kg of dry soil had a cadmium concentration in the dry carpophores of 2.1 to 7.5 mg/kg, which gives a concentration factor of 13 to 47 (Stijve and Besson 1976).

Red maple and white pine grown on soils containing 1.0 mg Cd/kg showed symptoms of cadmium poisoning. The cadmium accumulation in the tissue was 33.4 mg/kg dry weight in the roots of red maple and 4.9 mg/kg dry weight in the roots of white pine (Mitchell and Fretz 1977).

A level of 50 mg Cd/kg of soil has been quoted as the threshold value, above which damage can occur to plants (Dugdale 1978).

In the region of Japan where the Itai-Itai disease was identified, a positive correlation was found between the incidence of the disease and cadmium concentration in the upper stratum of the soil in the rice fields. The cadmium concentration of soil ranged between 0.9 and 3.0 mg/kg (Friberg *et al.* 1974, p. 154), and the concentration of cadmium in the rice averaged 0.68 mg/kg or about 10 times higher than in other parts of Japan (0.066 mg/kg) (Friberg *et al.* 1974, p. 139). The water used for irrigation had a maximum concentration of 0.009 mg/L of cadmium. The drainage from the mine suspected of contaminating the irrigation waters had much higher concentrations. The concentration of cadmium in the suspended materials was also quite high, and it is the cadmium in these suspended

materials that is suspected of contaminating the rice (Friberg *et al.* 1974, pp. 155-156).

It is interesting to note that good irrigation practice with sewage effluent and effluent-sludge mixture containing low levels of cadmium can lead to a decrease in the cadmium concentration of plants owing to an increased biomass (Sidle and Sopper 1976).

Recommended Objective for Irrigation Water

The accumulation of cadmium in plant tissues poses a very serious risk to human health. This, and not the damage to the plant itself, is the determining factor in establishing an objective for cadmium concentration in irrigation waters. It is recommended that the maximum concentration of total cadmium in irrigation water be 0.01 mg/L. This objective is based on total, i.e., dissolved plus suspended, cadmium. It should be noted that irrigation water is only one of the vectors that contribute to the accumulation of cadmium in soils. Two other important sources of cadmium are atmospheric deposition and the use of phosphate and superphosphate fertilizers.

Recreation and Aesthetics

Potable water supplies in recreational areas should comply with the level of 0.01 mg/L total cadmium for raw public water supplies. The level recommended for aquatic life, 0.0002 mg/L, is much lower and will ensure the safety of any water used for contact sports.

Recommended Objective for Recreational Waters

The recommended objective for cadmium in natural waters used for recreational purposes is 0.01 mg/L total cadmium.

Industrial Water Supplies

The only industry in which the concentration of cadmium in the process water is of importance is the food industry.

The general principle is that water which is incorporated in the final product should meet the same requirements as those for drinking water.

Recommended Objective for Industrial Water Supplies

An objective of 0.01 mg/L of total cadmium is recommended for water which is incorporated in the final product in the food industry. This includes the water used for cooling cans in the canning industry.

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